Amendments to the Claims:

This listing of claims will replace all prior versions, and listings, of claims in the application.

Listing of Claims:

Claims 1-18 (canceled).

Claim 1 (previously presented) A method for treatment and/or prophylaxis of inflammation in a mammalian patient, which method comprises administering to said patient an effective amount of apoptotic bodies, wherein said apoptotic bodies exhibit at least two characteristics comprising DNA fragmentation, surface exposure of phosphatidylserine, or altered mitochondrial membrane permeability, to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of inflammation in the treated patient.

Claim 2 (previously presented) The method of claim 19, wherein the apoptotic bodies are in a liquid suspension along with viable cells.

Claim 3 (previously presented) The method of claim 20, wherein the apoptotic bodies comprise from 10% to 90% of the cellular portion of the suspension.

Claim 4 (previously presented) The method of claim 21, wherein the apoptotic bodies comprise from 30% to 70% of the cellular portion of the suspension.

Claim 5 (previously presented) The method of claim 19, wherein the apoptotic bodies are derived from extracorporeal treatment of blood cells compatible with those of the mammalian patient.

Claim 24 (currently amended) ~~The method of claim 19,~~ A method for treatment and/or prophylaxis of inflammation in a mammalian patient, which method comprises administering to said patient an effective amount of apoptotic bodies, wherein ~~the~~ said apoptotic bodies are derived from established cultured cell lines and exhibit at least two characteristics comprising DNA fragmentation, surface exposure of phosphatidylserine, or altered mitochondrial membrane permeability, to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of inflammation in the treated patient.

Claim 25 (previously presented) The method of claim 23, wherein the blood cells are white blood cells of blood compatible with that of the mammalian patient.

Claim 26 (previously presented) The method of claim 25, wherein the blood cells are the patient's own white blood cells.

Claim 27 (previously presented) The method of claim 26, wherein the blood cells are the patient's own T lymphocytes.

Claim 28 (previously presented) The method of claim 19, further comprising administering to a human patient a dosage of apoptotic bodies comprising from 10,000 to 10,000,000 apoptotic bodies per kilogram body weight of the patient.

Claim 29 (previously presented) The method of claim 28, wherein the dosage contains from 500,000 to 5,000,000 apoptotic bodies per kilogram body weight of the patient.

Claim 30 (previously presented) The method of claim 28, wherein the dosage contains from 1,500,000 to 4,000,000 apoptotic bodies per kilogram body weight of the patient.

Claims 31-45. (Canceled).

Claim 46 (previously presented) A method for reducing and/or preventing an inflammatory component of a disease in a mammalian patient which method comprises

administering to said mammalian patient an effective amount of apoptotic bodies to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of inflammation in the patient, and wherein said apoptotic bodies exhibit at least two characteristics comprising DNA fragmentation, surface exposure of phosphatidylserine, or altered mitochondrial membrane permeability.

Claim 47 (previously presented) The method of claim 46, wherein the apoptotic bodies are in a liquid suspension along with viable cells.

Claim 48 (previously presented) The method of claim 47, wherein the apoptotic bodies comprise from 10% to 90% of the cellular portion of the suspension.

Claim 49 (previously presented) The method of claim 48, wherein the apoptotic bodies comprise from 30% to 70% of the cellular portion of the suspension.

Claim 50 (previously presented) The method of claim 46, wherein the apoptotic bodies are derived from extracorporeal treatment of blood cells compatible with those of the mammalian patient.

Claim 51 (currently amended) ~~The method of claim 46,~~ A method for reducing and/or preventing an inflammatory component of a disease in a mammalian patient which method comprises administering to said mammalian patient an effective amount of apoptotic bodies to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of inflammation in the patient, and wherein said apoptotic bodies wherein the apoptotic bodies are derived from established cultured cell lines and exhibit at least two characteristics comprising DNA fragmentation, surface exposure of phosphatidylserine, or altered mitochondrial membrane permeability.

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Claim 52 (previously presented) The method of claim 50, wherein the blood cells are white blood cells of blood compatible with that of the mammalian patient.

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Claim 53 (previously presented) The method of claim 52, wherein the blood cells are the patient's own white blood cells.

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Claim 54 (previously presented) The method of claim 53, wherein the blood cells are the patient's own T lymphocytes.

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Claim 55 (previously presented) The method of claim 46, further comprising administering to a human patient a dosage of apoptotic bodies comprising from 10,000 to 10,000,000 apoptotic bodies per kilogram body weight of the patient.

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Claim 56 (previously presented) The method of claim 55, wherein the dosage contains from 500,000 to 5,000,000 apoptotic bodies per kilogram body weight of the patient.

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Claim 57 (previously presented) The method of claim 55, wherein the dosage contains from 1,500,000 to 4,000,000 apoptotic bodies per kilogram body weight of the patient.

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Claim 58 (previously presented) A method for reducing and/or preventing inflammation in a mammalian patient, which method comprises administering to said patient an effective amount of apoptotic bodies, wherein said apoptotic bodies exhibit at least two characteristics comprising the binding of Fas ligands to Fas receptors, caspase activation, DNA fragmentation, surface exposure of phosphatidylserine, altered mitochondrial membrane permeability, or release of mitochondrial cytochrome-c, to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing and/or preventing inflammation in the treated patient.

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Claim 59 (previously presented) A method for reducing inflammation in a mammalian patient with an inflammatory disorder, which method comprises administering to said mammalian patient an effective amount of apoptotic bodies to up-regulate the *in vivo* generation

of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of-inflammation in the patient, wherein said apoptotic bodies exhibit at least two characteristics comprising the binding of Fas ligands to Fas receptors, caspase activation, DNA fragmentation, surface exposure of phosphatidylserine, altered mitochondrial membrane permeability, or release of mitochondrial cytochrome-c.